Journal of Forensic and Legal Medicine 19 (2012) 363

Contents lists available at SciVerse ScienceDirect

Journal of Forensic and Legal Medicine

journal homepage: www.elsevier.com/locate/jflm

Letter to the Editor

Can mild organophosphate poisoning result in myocardial infarction?

Dear Editor,

I read with interest the case presented by Karasu-Minareci et al.[1] recently published in your journal. The authors have reported a 52-year-old male smoker with accidental ingestion of an unknown amount of parathion presenting to their emergency department about an hour after the ingestion. At presentation, he had chest pain, dizziness, myalgia, vomiting, increased salivation and diaphoresis without any specific neurological symptoms and muscle weakness. His blood pressure was 110/70 mmHg and his heart rate was 80 beats/min. Also, he had hyperglycemia, leukocytosis, mild anemia, hyponatremia, and increased cardiac markers at presentation. His electrocardiogram revealed acute inferior wall myocardial infarction (MI). In the coronary angiography performed, 90-percent stenosis of the right coronary artery was demonstrated. However, serum cholinesterase level could not be measured because of the insufficient laboratory conditions. Interestingly, nothing has been performed for the management of organophosphate (OP) poisoning. The authors have declared that vomiting, increased salivation, and diaphoresis had been due to OP poisoning and have correlated his MI to mechanisms including coronary vasoconstriction due to acetylcholine, vulnerability of the patient to stress and intensive exercise due to exposure to OP!, release of catecholamines and vasoactive amines due to induction of stress by pesticides, and hypersensitivity/inflammatory reaction in the type II Kounis syndrome (because the patient had leukocytosis!).

The major concern about this study is that was the patient's MI really due to mild OP poisoning? It seems that even the diagnosis of OP poisoning is questionable in this patient, especially because no confirmatory lab test is present to confirm such diagnosis. As you know, chest pain, dizziness, vomiting, and diaphoresis can all be due to MI.[2] The only positive points in favor of OP poisoning in this patient are increased salivation and maybe, his normal blood pressure and heart rate. Since we usually expect the patient to experience hypotension and sinus bradycardia in the early cholinergic phase of the OP poisoning (due to the stimulation of muscarinic receptors)[3] and also up to one-half with inferior wall MI show evidence of parasympathetic hyperactivity (bradycardia and/or hypotension),[2] the patient's normal blood pressure and heart rate may be due to the stimulation of the nicotinic receptors which may be seen in OP poisoning.[3]

Is it possible that increased acetylcholine results in coronary vasoconstriction while inducing such mild symptoms even without the need for treatment with atropine and pralidoxime? To my best knowledge, only one case report of MI due to mild OP poisoning has been reported in the literature (the patient only had drowsiness) which has happened seven days after the acute poisoning. It seems that even in this recent case, MI had not been due to OP poisoning in spite of all explanations the authors have offered.[4] This is because the studies have shown that MI usually happens within the first few hours of OP poisoning and in accompany with other apparent cholinergic signs and symptoms in cases of severe poisoning. Hypoxemia, acidosis, and electrolyte derangements are major predisposing factors for the development of such cardiac complication.[5]

It seems that the patient could have developed MI because of fear, excitement, stress, and anger on the basis of a 90-percent coronary stenosis due to the release of the catecholamines.[6] I think the fear, stress, and excitement caused by the accidental ingestion of OP can best explain the development of MI in this patient. In my opinion, explanations such as hypersensitivity/inflammatory reaction in the type II Kounis syndrome in this patient with only a leukocytosis without clinical signs and symptoms of hypersensitivity, cannot show the role of mild OP poisoning in the development of MI. As already known, MI can induce leukocytosis and hyperglycemia because of the stress that causes.[2] In addition, I calculate that this case would have a Naranjo score of 1.[7] It therefore seems that mild organophosphate poisoning cannot result in myocardial infarction.

Conflicts of interest

None.

References

1. Karasu-Minareci E, Gunay N, Minareci K, Sadan G, Ozbey G. What may be happen after an organophosphate exposure: acute myocardial infarction? *J Forensic Leg Med* 2012;**19**:94–6.
2. Antman EM, Loscalzo J. ST-segment elevation myocardial infarction. In: Longo D, Fauci A, Kasper D, Hauser S, Jameson JL, Loscalzo J, editors. *Harrison's principles of internal medicine*. 18th ed. New York: McGraw-Hill; 2012. p. 2021–35.
3. Insecticides: organic phosphorus compounds and carbamates. In: Hoffman RS, Nelson LS, Howland MA, Lewin NA, Flomenbaum NE, Goldfrank LR, editors. *Goldfrank's manual of toxicologic emergencies*. 1st ed. New York: McGraw-Hill; 2007. p. 837–42.
4. Kidiyoor Y, Nayak VC, Devi V, Bakkannavar SM, Kumar GP, Menezes RG. A rare case of myocardial infarction due to parathion poisoning. *J Forensic Leg Med* 2009;**16**:472–4.
5. Saadeh AM, Farrakhan M, Ali-Ali MK. Cardiac manifestations of acute carbamate and organophosphorus poisoning. *Heart* 1997;**77**:461–4.
6. Vita JA, Treasure CB, Yeung AC, Vekshtein VI, Fantasia GM, Fish RD, et al. Patients with evidence of coronary endothelial dysfunction as assessed by acetylcholine infusion demonstrate marked increase in sensitivity to constrictor effects of catecholamines. *Circulation* 1992;**85**:1390–7.
7. Naranjo CA, Busto U, Sellers EM, Sandor P, Ruiz I, Roberts EA, Janecek E, Domecq C, Greenblatt DJ. A method for estimating the probability of adverse drug reactions. *Clin Pharmacol Ther* 1981;**30**:239–45.

Hossein Sanaei-Zadeh, MD, Associate Professor
Department of Forensic Medicine and Toxicology, Tehran University of Medical Sciences, Tehran, Iran
E-mail address: h-sanaiezadeh@tums.ac.ir

20 December 2011
Available online 4 March 2012

doi:10.1016/j.jflm.2012.02.030